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TRANSACTIONS

OF THE

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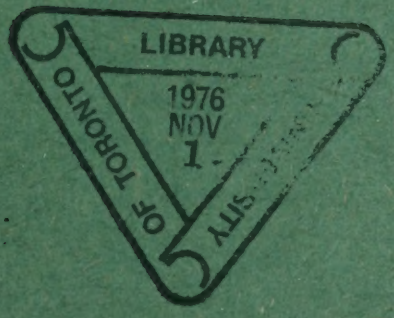

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JANUARY, 1916.

VOLUME IX. No. 3.

Proceedings of a Meeting of the Society held on Friday, December 17th, 1915, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W., Dr. F. M. SANDWITH, F.R.C.P., *President*, in the Chair.

AN INTERESTING CASE OF EOSINOPHILIA.

BY

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Lecturer, London School of Tropical Medicine.
Assistant Physician Seamen's Hospital, Albert Docks, etc.

The usual causes given for an increase of the eosinophile cells in the circulating blood are :—

(1) Helminthiasis.
(2) Acute and Chronic Skin Diseases.
(3) Malignant Bone Tumours.
(4) Asthma.
(5) After Injections of Tuberculin.
(6) Splenomedullary Leukaemia.
(7) Undetermined Causes.

Recently, I saw a patient from India, said to be suffering from slight fever. He looked sallow, and it was this, as much as anything, that brought him before me for examination. His conjunctiva was a good colour and did not indicate that he had anæmia. Apart from having many pigmented moles on his body generally, and a peculiar pigmented condition of the skin, he was organically sound.

An examination of his blood gave the following :—

Reds	4,410,000
Whites	10,400
Hæmoglobin	95 per cent.

Differential :—

	No. Counted.	Per Cent.	No. per c.mm.
Polymorphonuclear ...	118	23·6	2,454·4
Large Mononuclear ...	13	2·6	270·4
Lymphocytes	121	24·2	2,516·8
Eosinophiles	244	48·8	5,075·2
Transitional	3	·6	62·4
Mast Cells	1	·2	20·8
	500	100·0	10,400·0

Films.—No parasites of any kind seen, no signs of recent malaria.

The blood count then shewed that the patient had an absolute and relative increase of eosinophiles of a very high degree. A specimen of fæces was therefore obtained, but a careful examination of this, both by Dr. WENYON and myself, revealed no abnormalities—no ova of any helminths were present, nor any amœbæ, flagellates, or other protozoal organisms.

The eosinophilia, the patient afterwards informed me, had already been detected in India, and the stools carefully examined there, the results being the same—namely, negative.

Day blood shewed no filarial embryos. I could not obtain the night blood ; but there were no signs of filariasis clinically, and I have never seen an eosinophilia of such a degree in that condition. The same may be said of hydatid disease. There was not at the examination, nor had there ever been, any evidence of guinea worm.

Trichinosis still remains as a possibility. Clinically there was no history of such an attack, apart from him having suffered from diarrhœa

for three months when he first went to India, thirteen years ago. He had never had any obscure attack of enteritis, neuralgia, or rheumatism, but did have a certain amount of fever during his stay in India. As this always responded to quinine it was probably malarial. Supposing that he had acquired trichinosis in India, would he still have such a high degree of eosinophilia so many years afterwards? The only way to be certain of this—and even then one might not be absolutely so—would be to cut out a piece of muscle and look for the embryos. The patient was not inclined to have this done, however; so one must, I suppose, apply the verdict "not proven" to the possibility of this disease. Apart from this, of the only other usually ascribed causes present, was the condition of the skin. This was somewhat peculiar, and consisted of many pigmented warts (20 or more) scattered over the body, five or six being on the nape of the neck. These he said he had had from childhood. They had not grown bigger lately and had never specially troubled him. It was once suggested to him in India that they might be removed by carbonic acid snow, but this was not carried out. In addition to the warts, there was a peculiar pigmented condition of the skin, linear pigmented marks distributed especially on the back. These were flat, not raised in any way, and shewed no sign of any inflammation or irritation. The skin did not itch, nor were there any marks of scratching. Pigmented moles or warts sometimes develop into melanotic sarcomas, and the possibility was duly considered, but as far as one could judge there was no evidence of such a change having taken place.

I have seen eosinophilia in several varieties of skin disease, but have never examined the blood of an exactly similar case to this before. I believe it possible, however, that the condition of the blood is really due to the skin lesions. Dr. WENYON, to whom I mentioned this case, told me that he saw a very extraordinary case of eosinophilia when he was in the Sudan in 1907, a short note of which appeared in the *Third Report of the Wellcome Research Laboratories* (p. 130). The percentage of eosinophiles reached 70 per cent. The patient was suffering from a chronic skin eruption on the lower half of the body. The eruption took the form of localised thickenings of the skin, causing it to feel hard and rough. There was much irritation, and the condition had lasted six or seven years. The stools were not examined and there are no details as to the possibility of trichinosis.

In constantly examining the blood of tropical cases not suffering from any definite disease, one is struck by the fact that a certain moderate degree of eosinophilia is not an uncommon occurrence. By that I mean without the presence of helminths, or any of the other recognised causes. Recently, I see a slight increase in the eosinophiles has been described in dysentery. This has probably nothing to do with that disease, but only indicates a mixed infection with one of the recognised causes, or with the type of case just mentioned above.

I remember a very interesting case of anæmia and eosinophiles in a patient in the Seamen's Hospital, Royal Albert Dock, when I was Superintendent at the London School of Tropical Medicine. The eosinophiles were always somewhere between 14 and 20 per cent., yet no cause could be found for this. The patient ultimately died, and, as I performed the autopsy upon him, I had the opportunity of carefully examining all the tissues of his body. No recognised cause of eosinophilia was present. There were no helminths of any kind, no skin disease, no bone disease or anything else to account for it. Having done a careful post-mortem, one could say this definitely, of course. Probably many other indefinite cases of a similar nature may exist, and it would certainly be of interest if anyone meeting such would carefully record them.

DETAILS OF CASE.

Mr. G., 38, male. November 14th, 1915.

Complaint.—Fever and indefinite symptoms.

History.—Went to India thirteen years ago. Went to Australia for his first leave after five years' service. Home for six weeks after eight years' service. Came home again on leave, July, 1914.

Previous illnesses.—Persistent diarrhœa for three months on going out first. Attacks of colic after that. No definite dysentery. No enteric. A fair amount of fever during his stay in India. This responded to quinine. Appendix removed November, 1914. Never venereal disease. Never rheumatism or muscular pains.

Present illness.—Felt chilly and feverish September, 1915. Temperature over 100° had a cold also, however. Took quinine up to grs. xx. a day, which cured it. After that up to November 4th, when he was seen, quite well.

Examination.—Sallow looking; skin looks pigmented; conjunctiva, however, a good colour; pupils react normally. Teeth—Plate in upper jaw; mucous membrane in floor of mouth seems hypertrophied under tongue at frœnum. Stripped moderately developed. Many pigmented moles over body, especially at back of neck. In addition to these, pigmented linear streaks on back. Systems:—Lungs healthy. Ditto heart. Liver 6, 7 and c. m. Spleen can just be felt under costal margin.

Good scar of old appendix operation. Nothing in groins, penis or legs. Urine: no albumin, blood or sugar. Fæces: no ova, no protozoa of any kind. Blood examination, see text. Diagnosis, ?

Discussion.

The President: The eosinophile leucocyte has been called the most capricious of the blood cells, and I dare say there may be occasions when eosinophilia is present besides those in the list which Dr. Low gives at the beginning of his paper. I notice he says pigmented warts were present. I presume there were no pustules, or they would have been mentioned. I am not prepared to give any diagnosis.

I notice that the Fellows are silent, so that it only remains for me to thank Dr. Low for bringing the case forward.

Dr. G. C. Low: In answer to Dr. Sandwith, I may say that there were no pustules, nor any inflammation of the warts. If I see the patient again I shall excise one of the warts and have it examined histologically, to see if this will throw any light on the condition.

INTESTINAL PARASITES IN NORTHERN SIAM.

BY

DR. W. F. J. KERR, CHIENGMAI, SIAM.

During the past year, I carried out an investigation to determine the relative frequency of infection with various intestinal parasites among the prisoners in Chiengmai jail.

A series of 230 adult male prisoners was examined. Of this number 74 per cent. were Laos, and 13 per cent. Shans and Karens, all natives of Northern Siam, most of whom had spent their whole lives in the Chiengmai district. The remainder were made up of small numbers of Siamese, Yunnanese, Indians, and Burmese. The Chiengmai district was, until the last two years, two or three weeks' journey from the nearest seaport; now an advancing railroad brings it within five or six days of Bangkok.

The routine followed in every case of this series was to make a microscopical examination of the motions, and then to give an anthelmintic.

For the microscopical examination very thin films of fæces were employed, always diluted with water, unless the motions were liquid; 21/26 mm. coverglasses were used, and the whole of each film gone through. In about 70 of the earlier cases a second film was only examined when no hookworm ova were found in the first; in all the other cases two films were examined. A laxative was given before the microscopical examination to ensure getting a motion, but the motion so obtained was usually formed.

On the evening before the anthelmintic, the cases were restricted to a liquid diet, and given 6 drachms of magnesium sulphate on going to bed. The anthelmintics used were: three 30-grain doses of β-naphthol in 27 cases;* the same doses of β-naphthol, with 2 grains of santonin in 5 cases; eucalyptus mixture in 63 cases; eucalyptus mixture and 2 grains of santonin in 135 cases. The anthelmintic was followed by another

* As recommended by BURTON NICOL. *Journal of Tropical Medicine and Hygiene.* Feb. 15, 1911.

dose of magnesium sulphate, except in some of the cases taking eucalyptus mixture when the motions were free.

The motions were collected for two to four hours after the last purgative; if they had been collected for 24 hours the number of parasites obtained would probably have been larger. From the motions so obtained the worms were recovered by the sedimentation method recommended by Major CLAYTON LANE.* Straining through a fine sieve was found to be unsatisfactory.

The following table gives the main results of the investigation in percentages :—

	Primary examination positive.	Parasites found after anthelmintic.	Parasites found after anthelmintic when primary examination negative.	Total infected.
Fannia sp. (?)	0·0	1·3	1·3	1·3
Tænia saginata, GOEZE	30·0	56·1	28·3	58·3
Opisthorchis felineus, RIV. (?)	17·0	0·0	0·0	17·0
Strongyloides stercoralis, BAV. ...	18·3	0·0	0·0	18·3
Ascaris lumbricoides, LINN.	46·5	25·7	11·7	58·2
Oxyuris vermicularis, LINN.	1·3	50·0	48·7	50·0
Agchylostoma sp.	(?)	14·8	(?)	14·8
Necator americanus, STILES	56·5	88·7	37·8	94·3
Trichocephalus trichiurus, RUD. ...	27·0	1·7	1·3	28·3
Entamœba histolytica, SCHAUD., cysts	0·9	0·0	0·0	0·9
Entamœba coli, SCHAUD., cysts ...	6·0	0·0	0·0	6·0
Entamœba sp., trophic stage	23·1	0·0	0·0	23·1
Lamblia intestinalis, BLANCH... ...	1·0	0·0	0·0	1·0
Trichomonas hominis, DAVAINE ...	2·6	0·0	0·0	2·6

In the above table the first column indicates the percentage found infected at the primary examination whatever stage of the parasite was found, not necessarily microscopic.

* *Indian Medical Gazette.* June, 1913.

The results given for the protozoa do not represent the full series of 230 cases, as the earlier cases were not examined sufficiently carefully for these organisms, and had to be rejected. The figures for *Lamblia* and *Trichomonas* represent an examination of 195 cases, for *Entamœba* of 117 cases.

Most of the prisoners harboured more than one species of parasite, as the table implies. If protozoa are excluded, 31 per cent. of the cases had three species of parasite, 30 per cent. had four, 14 per cent. had two, 13 per cent. had five, 7 per cent. had one, and 4 per cent. had six; there was a single case with seven species, and a single case, an Indian, where no parasites were found.

The combinations of the parasites were such as might be expected from the relative frequency of the infections. The prisoner with seven species was a Lao who had never been outside the Chiengmai district; he harboured *Necator americanus*, *Agchylostoma ceylanicum*, *Ascaris lumbricoides*, *Trichocephalus trichiurus*, *Opisthorchis felineus*, *Tænia saginata*, and *Oxyuris vermicularis*. Five of these species were recovered after the anthelmintic, including 39 *Necator* and 11 *Tænia*; *Trichocephalus* and *Opisthorchis* were not recovered.

The following are further details of the various parasites found:—

Fannia sp. (?).—A maggot with feathery processes on the segments was found in three cases after the anthelmintic; they probably belonged to this genus, and were too big to have been an infection after the motions were passed.

Tænia saginata.—This cestode was very common; if the cases where only segments were found are put down as having only one worm each, all the infected cases had an average of 2·5 worms each. Heads were recovered in 74 cases, the average number of heads from each being 3·5. From one man no less than 14 heads were obtained, from another 13, and from three others 11. On one occasion 16 heads were removed post-mortem from a prisoner. In about 17 per cent. of the infected cases oncospheres were found at the primary examination. In five instances no segments were found after the anthelmintic, though they had been seen at the preliminary examination; these men stated that large masses of tapeworm came away with the purge on the evening before the anthelmintic; it is possible that in some of them the heads came away first—this occurred in a case of mine outside the jail.

While it is not surprising that infection with *Tænia saginata* is common, as various forms of uncooked beef are much eaten by the natives, it is remarkable that *Tænia solium* was not found, as raw pork is eaten even more than raw beef. A similar scarcity of *Tænia solium* is reported from the Philippines.

In this series no other species of cestode was recognised at all, but in an official recently arrived from Bangkok I found cestode oncospheres resembling figures of those of *Hymenolepis nana*. Unfortunately, the adult parasites were not obtained, as no proper examination was made of the motion after giving eucalyptus mixture.

Opisthorchis felineus (?)—In thirty-nine cases of the series, 17 per cent., a very small fluke egg was found at the preliminary examination in scanty numbers. This egg measured about ·027/·016 mm. and had a small boss at the pole opposite the operculum, agreeing in these and other particulars with the egg of *Opisthorchis sinensis*, which it was at first thought to be; but this identification could not then be put to the test, as no flukes were recovered after the anthelmintic in this series. Later, through the kindness of Dr. C. W. Mason, I was present at the post-mortem of a man in whom these ova had been found in the motions in large numbers during the life; some thousands of small flukes were obtained from the liver and gall-bladder. These parasites agreed fairly well with figures and descriptions of *Opisthorchis felineus*. After this I myself had a post-mortem on a prisoner in whose motions these ova had been found a month previously, no anthelmintic having been given on account of the patient's state of health; there were fourteen flukes of the same species in the liver and gall-bladder. In about half the infected cases there was some tenderness to pressure over the liver, chiefly in the gall-bladder region, in no cases marked; some of the cases complained of obscure abdominal pains, while others shewed no symptoms.

Though no fluke was obtained in this series, a second species was obtained later from a prisoner after the administration of eucalyptus mixture; this fluke I was unable to identify, and have forwarded to Dr. Leiper for determination.

Ascaris lumbricoides.—It will be seen from the table that this worm was recovered after an anthelmintic in less than half the infected cases; in the first ninety cases, where no santonin was given, worms were only recovered from ten out of fifty-six infections. The average number

of worms collected from each infected case was 1·5, the largest number obtained from one case being eight. In children the infection rate is probably higher, and the number of worms per individual much larger, than among adults. I have seen immense tangled masses of these worms recovered from children; one of my patients, a girl 3½ years old, passed 200 of these worms in three days. Sometimes in children *Ascaris* gives rise to symptoms suggesting serious abdominal lesions; in one case I diagnosed acute appendicitis, in another intussusception; the latter was confirmed by a surgeon, who recommended operation; in both these cases the symptoms cleared up at once after passing numbers of worms.

Oxyuris vermicularis.—The ova of this parasite were only found once, at the preliminary examination, and adult parasites twice. The numbers obtained after the anthelmintic were usually small, about six or seven, but on two or three occasions the motions were seething with them.

Agchylostoma sp.—Worms belonging to this genus were found in thirty-four cases of the series, 14·8 per cent., usually in small numbers. From one case eleven worms were recovered, and from another nine; from no other case were more than two worms recovered, the average for all infected cases being 1·8 worms. No attempt was made to distinguish the ova of *Agchylostoma* from those of *Necator* at the preliminary examination, all hookworm ova being put down as *Necator*; this has made no difference to the final result, as in only three cases was *Agchylostoma* without *Necator* recovered after an anthelmintic, and in none of these cases had hookworm ova been found at the preliminary examination.

These worms were at first thought to be *Agchylostoma duodenale;* later it was noticed that they were not typical of that species, and all those kept—38 out of the original 61, some having been sent to Dr. Leiper and others lost—were gone through again. Of the 38 worms, 33 were found to be *A. ceylanicum,* three *A. duodenale,* and two were too badly preserved to form an opinion on; the points relied on in making the distinction between the two species being the very small median teeth and the form of the dorsal bursal ray in *A. ceylanicum.*

Necator americanus.—If the stools had been collected for twenty-four hours after the anthelmintic, instead of only the first three or four stools,

the infection rate for *Necator* in this series would have been found higher than 94 per cent.; probably every case was infected.

In thirteen cases ova were found at the preliminary examination, when no hookworms were recovered after the anthelmintic; it is improbable that these were ova of *Strongyloides stercoralis*, which are rarely found in the stools; in addition, in only two of these cases was the embryo of *Strongyloides* found.

Other investigations made in Northern Siam also shew a high rate of hookworm infection. Dr. W. H. Beach, of Nan, found an infection rate of 85 per cent in over 200 cases, mostly children. Dr. W. J. W. McKean, of Chiengmai, examined 242 cases and found 54 per cent. infected; 216 of these were children with an average age of fourteen. Dr. Beach kindly sent me two worms which were both *Necator americanus*, and Dr. McKean was good enough to give me the opportunity of going over 274 hookworms from his cases; 273 of these were *Necator americanus*, and one, rather damaged, was an *Agchylostoma*, apparently *A. ceylanicum*. There can be no doubt that *Necator americanus* is the common hookworm throughout Northern Siam, and is indigenous to the country, as it is in neighbouring territories.

The average number of *Necators* obtained from each infected case was 22, the largest number obtained from one case being 169.

The hæmoglobin index of 200 cases was estimated with a tinted hæmoglobinometer. While the results given by such an instrument cannot be considered altogether reliable, it may be worth while giving them :—

13	cases with	no	hookworms	had an	average of	90% hæmoglobin.
84	,,	1-10	,,	,,	,,	93% ,,
47	,,	10-25	,,	,,	,,	90% ,,
38	,,	25-50	,,	,,	,,	88% ,,
11	,,	50-100	,,	,,	,,	90% ,,
5	,,	100-150	,,	,,	,,	84% ,,
2	,,	150-200	,,	,,	,,	87% ,,

It is unlikely that any considerable degree of anæmia would result from the small numbers of hookworm found; chronic malaria was probably the chief factor in causing what anæmia there was in this series.

Lamblia intestinalis.—This species was found in two cases of the

series; in one the motions were formed, in the other diarrhœic. I have seen this organism in two civilian patients associated with dysenteric stools; no entamœbæ were found, and in both the *Lamblia* disappeared and the stools cleared up on treatment with β-naphthol.

Trichomonas hominis.—This flagellate was found in five cases. It is very common in loose stools.

Entamœba sp.—Entamœbæ were carefully searched for in 117 cases; among these cysts of *E. coli* were found in seven cases, while those of *E. histolytica* were found in only one; they were plentiful in this case, but mixed with *E. coli* cysts. It is possible that the small number of cases infected with cysts was due to the fact that a laxative was given before the examination, though the motion so obtained was usually formed, or half-formed. Nothing was counted a cyst unless the spores could be distinctly counted. As WALKER has pointed out,* there is another organism, *Blastocytis hominis (?)*, which is apt to be mistaken for an entamœbic cyst; this organism was present in 47 per cent. of the cases examined.

Entamœbæ in the trophic stage were found in 27 cases; they all had the heavy chromatin ring and dense protoplasm, said to be typical of *E. coli*, but they varied greatly in size; some of them may have been the pre-encysted stage of *E. histolytica*.

In cases where there was doubt about entamœbæ or cysts, stained films were made; the method of staining followed was that recommended by WALKER[1], a wet hæmatoxylin method, which gave excellent results with the minimum of trouble.

The anthelmintics used.—As already stated, the anthelmintics used were eucalyptus mixture and β-naphthol, either alone or combined with santonin. Without the santonin neither were of much effect in expelling round worms. The average number of hookworms obtained after eucalyptus mixture was somewhat larger than after β-naphthol; with the former they were passed alive, while with the latter they were dead. The proglottides of *Tænia* after β-naphthol came away separately, or in very short chains, while the heads were rarely found; after eucalyptus mixture the proglottides were in long chains, and heads were frequently found.

[1] WALKER, *loc. cit.*

In this series one death occurred after the anthelmintic, a case of blackwater fever with hæmoglobinuria, bilious vomiting, and jaundice: fever came on within 24 hours of the administration of the anthelmintic, and was fatal in 48 hours. Cases of blackwater fever were occurring at that time among civilians outside the jail, and this death was not at first connected with the anthelmintic. After the completion of this series the treatment of prisoners for worms was still carried on with new arrivals, and among these were two more fatal cases of blackwater fever. These two cases occurred together; in both the symptoms came on within 24 hours of taking the anthelmintic, β-naphthol in both, and death supervened 24 hours later in one, 36 hours in the other. All three of these cases had had frequent attacks of fever, as most natives here have had, and two of them had very large spleens. I have no doubt in my own mind that the anthelmintic was the exciting cause of the attack in all three cases.

I have another untoward event following the administration of an anthelmintic to record. I was asked to see an official's wife who had been unwell for some time with low fever and some diarrhœa. When I saw her she was extremely anæmic, and her temperature was 99° F. An examination of the stools revealed hookworm ova in abundance—in larger numbers than I had ever seen before. Thinking the hookworm might be the cause of her condition, I gave her eucalyptus mixture, but, owing to her weak state, only in half doses. The result of this treatment on the hookworms is not known, as the motions were thrown away. However, within twelve hours her temperature began to go up, and she went safely through a typical relapse of typhoid, without doubt brought on by the anthelmintic.

NOTE.—Since the above was written, Lieutenant-Colonel LEIPER, R.A.M.C., has published a paper, "Notes of the Occurrence of Parasites presumably rare in Man," in the *Journal of the Royal Army Medical Corps* for June, 1915, wherein he refers to some of the parasites sent from Chiengmai, confirming my determination of *Agchylostoma ceylanicum*, LOOS, and identifying my undetermined fluke as *Euparyphium malayanum*, LEIPER, and my *Opisthorchis felineus* ♀ as *Opisthorchis viverrini*, POIRIER. W. F. J. K.

Dr. C. MORLEY WENYON: I wish to shew two specimens illustrating Coccidia which I have found in cases I have examined recently. During the last two months I have been examining, for protozoal infection, fæces of men invalided from Gallipoli for dysentery or other infection of the gut. I should like to take this opportunity of thanking Dr. G C. LOW for his kindness in assisting me in the matter, and for undertaking the examinations when I have been out of town. Dr. LOW's assistance has greatly increased the accuracy and value of these observations.

I may say at once that in connection with this work I have examined about 1000 specimens from over 500 different individuals, and out of that total I found that half of them have some form of protozoal infection. The most important point is that out of these 500 individuals, 10 per cent. are carriers of *Entamœba histolytica*. Perhaps I may be excused if I explain the method I adopt, because at the present time this is a very important matter. In making the diagnosis, I rely chiefly on the presence of cysts. The stool on these occasions may be normal, or nearly normal, and may contain no blood or mucus, so that we can only recognise the infection by finding some typical form, generally the encysted form, of *E. histolytica* in the stools. These encysted forms of the entamœbæ really correspond to the eggs of worms, for the finding of encysted forms of *E. histolytica* in the fæces is just as indicative of infection of the gut with this entamœba, as the finding of an ankylostome egg is of an infection of the gut with ankylostoma.

The cysts of *E. histolytica* are quite definite structures, and can be distinguished from the cysts of *E. coli* if one has had some practice in their examination. The important point is that the cysts always breed true, so to speak. If a man commences passing *E. coli* cysts he will do so for many years, and they never transform into cysts of *E. histolytica*; and similarly, if a man passes *E. histolytica* cysts, these again will never transform into cysts of *E. coli*.

The men who are carriers of *E. histolytica* are very dangerous, not only to themselves but also to other people. As an illustration of this danger, I may mention a case I examined some years ago for Professor SIMPSON. The man had a tremendous infection of *E. histolytica*. The infection was identified by finding large numbers of the characteristic cysts in his fæces. In spite of advice he started abroad, and at Port Said

he had a relapse in the form of an acute attack of dysentery, followed by liver abscess, from which he died. Such a case would have been saved if treated with emetine. Another case of the kind was a lady missionary returned from India. I found her to be passing large numbers of *E. histolytica* cysts. She had no dysentery at the time, but by means of her histolytica cysts I was able to produce an infection in cats, and I was able to pass the infection on from one cat to another. All these cats had typical amœbic ulcers of the gut. Eventually one cat developed amœbic abscess of the liver, and thus, starting from a carrier case, one had produced in cats the whole picture of the amœbic infection in man, with its complication of liver abscess.

The other day, I saw an interesting case of a man who had contracted amœbic dysentery, though he had never left England. He was working in the Docks on a transport. He was admitted into the London Hospital with an attack of dysentery. This cleared up. Soon after he was found to have liver abscess. This was opened and drained, and I found *E. histolytica* in the pus. I examined the stool, and found nothing on the first occasion. I again failed on the second examination, but on the third occasion I found typical cysts of *E. histolytica* in large numbers. That man was infected with *E. histolytica* in this country, very likely from a man coming home from abroad and carrying the infection. Unfortunately he died.

Of all the Gallipoli cases I have examined, 10 per cent of the 500, *i.e.*, 50 of them, are carrying *E. histolytica* infection. These men are dangerous to themselves, for they may relapse and get liver abscess. They may go back to a country like Gallipoli, where the conditions of spread of this disease are favourable, and they may be the means of spreading amœbic dysentery out there. These men ought, if possible, to be identified as carriers, and should be treated with emetine.

Other protozoal infections are not so important. Fifty per cent. are carrying *E. coli*, and a smaller number one or other of the flagellates. It is interesting to note that two parasites, which we know little of as regards their presence in man, have turned up in these Gallipoli cases. Dr. Woodcock identified a structure in a man at St. George's Hospital as a coccidium, and I was able to demonstrate that it belonged to the genus Isospora. Last night I was examining specimens, and I came across a structure which proved to be a totally different coccidium, belonging to

the genus Eimeria. One common coccidium of this genus is parasitic in the liver of rabbits, but the one I am shewing to-night is like the coccidium of the mouse intestine. The Isospora has been found in about ten out of the 500 cases I have examined. The other I only found last night in one case. Infection with coccidium is spread by individuals eating the oöcysts after they have completed their development on the ground. Of the type of infection they produce we really know nothing at present.

Dr G. C. Low: I think one point of special interest occurs in Dr. KERR's paper, and that is the toxic effects of the β-naphthol in some of the cases.

The drug might, of course, have acted as the exciting agent of a real attack of blackwater fever, but it seems much more likely that the hæmoglobinuria had nothing to do with blackwater fever, the condition rather being due directly to the naphthol. I recently gave a man three 20-grain doses of β-naphthol, and this produced toxic symptoms, namely, lethargy, a semi-comatose condition, and also spitting of blood. These conditions, alarming while they lasted, passed off on the second day, and the patient made a perfect recovery. There was never albumin in the urine, and the kidneys seemed to escape entirely. If one uses these large doses, namely, three 30-grain doses, as recommended by BURTON NICOL, then in susceptible individuals we must expect sometimes to get toxic symptoms.

As regards the case of amœbic dysentery and liver abscess, mentioned by Dr. WENYON, in a man who had never been out of England, such cases have been reported before. Major MARSHALL reported one in a man near Edinburgh, who was supposed to have got his infection from a soldier who had returned from India, and SAUNDBY and MILLER have also reported one from Birmingham. Such cases are of great interest, and shew that if amœbic carriers are introduced into England there is a possibility of them spreading dysentery amongst the indigenous population.

Miss MURIEL ROBERTSON: I should like to ask what evidence there is of treating amœbic carriers with emetine, and whether it is successful in getting rid of the *E. histolytica* cysts or not? The literature on the subject draws attention to the fact that cysts are relatively resistant

to emetine, and I would like to know if there is any clinical experience as to how far carriers, as apart from men suffering from acute attacks, can be treated with emetine.

Dr. G. C. Low: The point raised by Miss Robertson is one that requires further elucidation. Certainly, in some cases, proper doses of emetine cause the disappearance of *E. histolytica* cysts from the stools. I do not see why they should not disappear, because if the drug kills the vegetative form from which they arise, then no more cysts can be produced. The condition is similar to that of the crescents in malaria. Quinine has no effect upon these *per se*, but by killing the young forms which produce the crescents the latter then ultimately disappear. It is possible that the cases described, where the cysts have resisted the emetine, have not had proper doses, or in some way or other the amœbæ producing them have not been killed by the emetine. However, the subject requires more work and careful study. I am following up this line of research in several cases at the present moment, and hope soon to publish some definite results.

Dr. Andrew Balfour: It may be of interest if I state that Captain Archibald, working at Lemnos, found in about 5 per cent. of the cases he examined that *Trichomonas intestinalis* was present. The pathogenicity of these flagellates is not definitely determined, and although found in 5 per cent. of cases of dysenteric diarrhœa, that is not to say that this number were suffering from trichonomiasis. I have, however, seen some cases in which there was a very definite form of diarrhœa. It is characterised by the patient passing bright yellow stools in large quantities, and may or may not be associated with colic. Such cases would certainly appear to be due to trichomonas infection.

I think Dr. Wenyon's results are very interesting indeed. When I came back from the East I was surprised to find that histolytica cysts had hardly been found at all, but I thought this was probably due to emetine being so universally employed in the Gallipoli Peninsula. Even so, it seemed to me curious that so few histolytica cysts were being discovered. I now think this was largely due to the fact that the men who were examining the stools in these cases were not sufficiently familiar with the parasite of amœbic dysentery. We all know that

Dr. WENYON is an expert in this line of work, and it is, therefore, interesting to note that when he started his examination he began to find that histolytica infection was present.

I think, as regards histolytica infection, the number of cases of true amœbic dysentery in Gallipoli has been very considerable. It must be 60 or 70 per cent. of all cases, although, doubtless, in many instances, there has been mixed bacillary and amœbic infection.

Turning again to trichomonas infection, the Commission, of which I am a member, had its attention directed to the question of water supply at Mudros. The water was brought from Egypt by ships, and taken to the shore in tank barges. Remembering the work of ESCOMEL in Peru, who apparently traced an outbreak of flagellate diarrhœa to trichomonas in certain water tanks that had not been cleaned out for a long time, the Commission had a look at the water-tanks at Mudros East, and found that in some of them a good deal of slime had collected, as it had not been possible to clean them out for some time. Samples of the slime were taken, and they were submitted to examination by Captain ARCHIBALD, who found a flagellate present, which he regarded as a trichomonas. The tanks were cleaned out, but with what results I am unfortunately unable to say.

Dr. R. P. COCKIN: With reference to Dr. LOW's remarks on the toxic symptoms observed after treatment with β-naphthol, it may be of interest to record that, in a series of 1,400 cases of ankylostomiasis treated in the West Indies, I met with five cases such as those to which Dr. KERR calls attention.

The urine in these cases was identical in appearance with that which one sees in cases of blackwater fever, but differed from it on spectroscopic examination; the spectrum shewn in β-naphthol poisoning being similar to, if not identical with, that which is observed in cases of hæmatoporphyrinuria.

On the appearance of this complication, the use of β-naphthol was at once stopped, and the patient was placed on drachm doses of ext. ergotæ liq. thrice daily. The urine resumed its normal colour in from 24-72 hours, and there was no mortality.

Upon resumption of the treatment for the expulsion of the worms,

I thought it advisable to use thymol, and in the five cases recorded this was done, without any return of the dark-coloured urine.

The PRESIDENT: I understand from Dr. COCKIN'S remarks that the chemical condition of the urine he saw was similar to that occurring after sulphonal poisoning. Fellows of this Society will recognise that you may poison a man, as seen by his renal excretion, but that he has not necessarily the symptoms of true blackwater fever. It is very difficult, in a country where malaria is present, to be sure which condition is present, and Dr. KERR says there were cases outside getting blackwater fever at the same time.

With regard to Miss ROBERTSON'S question, I think that our rule in this country—in fact, in every country, I should say—is, having found evidence of amœbic dysentery, to give emetine. That must be the clinical rule until we know something better. Exceptions are possible where the patient has had large doses of emetine already, which you may or may not continue. I would remind Miss ROBERTSON that every case that travels from one country to another—say, from Gallipoli to Egypt or to England—where cysts are discovered in the fæces, is a carrier. That is probably the opinion of every Fellow in this room, though it is not the opinion of the whole of the profession generally. Many would say if you had an attack a month ago that it is all over. The carriers after an attack of dysentery are the people from whose fæces cysts can be obtained. Emetine will get rid of amœbæ if you go on long enough, and by the intracellular method. It is probably not so good by the mouth, as I do not think you can get at the organisms inside the bowel by giving it by this route. I agree with Dr. LOW that we have not had enough cases yet to be perfectly certain how many of these carrying cysts can be cured by prolonged doses of emetine. We do not know how many doses of emetine you are to give, nor the exact dose in grains, for such a purpose.

I am told there is a paper coming out to-night in the *British Medical Journal*, by Dr. DALE, which states that emetine may be a dangerous drug when given in ordinary doses over a prolonged period to cats and rabbits. With humans I have not yet met with signs of poisoning when 1-grain doses were given, nor have I noted any cumulative effects. I have

seen cases published where emetine in 1-grain doses was taken for three months at a time, with no untoward results.

I should like to congratulate Dr. WENYON on the work he has been doing.

We are in a condition of some ignorance about this Dardanelles type of dysentery, and the first thing to do is to find out by means of examining the fæces what organisms are present.

Another point of interest is to know how the flagellates and amœbæ get into the body, as, once being certain of this, we can then take the necessary steps to prevent them, and so diminish the disease.

THE CAUSATION OF SO-CALLED TROPICAL ANÆMIA.

BY

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Every one with tropical experience will agree that Europeans, who have lived long in the tropics, usually become ænemic-*looking*. At one time it was usually tacitly assumed that this appearance denoted true anæmia, *i.e.*, an amount of hæmoglobin in the blood below the normal. Estimates of the amount of hæmoglobin in the blood do not, however, support this view. The amount of hæmoglobin, if reduced at all in the tropics, is not reduced sufficiently to give rise to a definite anæmia.

It is sometimes suggested that the anæmic appearance is due to ischæmia of the skin capillaries,* *i.e.*, to a contraction or diminution in the number of the skin capillaries causing a diminution of the quantity of blood in the skin. I am not aware that this ischæmia has ever been demonstrated, or that there is any independent support for this view. On *a priori* grounds it is very unlikely that there is a relative absence of blood in the tropics. It is undoubted that the chief skin function, sweating, is more marked in the tropics; and it is unlikely that the blood supply to the skin diminishes with this increased functional activity. It is a general physiological rule that the blood supply to an organ increases when the organ is functionating. Moreover, it is generally held, and I think quite correctly, that the heat regulating mechanism requires that in the tropics the cutaneous vessels shall be dilated.†

The cause of a person's skin having a particular colour, is by no means such a simple matter as it may appear at first sight. Before any scientific discussion on skin colour can take place, it is necessary to clearly understand the optical factors which influence the colour.

From the optical point of view, the skin consists of three layers: —

1. The unpigmented outer epidermis, more or less transparent.

* *Manual of Tropical Medicine*. CASTELLANI & CHALMERS. 1913. p. 101

† *Ibid.*, p. 84.

2. The pigmented layer more or less absent in the case of Europeans living in a temperate climate.

3. The true skin with its blood vessels.

Again, from the optical point of view, ordinary white light really consists of a mixture of light of all colours of the spectrum, and the colour of an object depends on the relative amounts of these different coloured lights which the object sends back to the eye of an observer. A white transparent object, such as glass, allows practically all the light to pass through unchanged. A white opaque object, such as chalk, scatters most of the light in all directions, and appears white because light of each colour is scattered to the same extent. If one colour is scattered to a greater extent than the other colours, the object appears tinged with that colour. It is by the light which is scattered by the various layers of the skin that we judge the colour of the skin. But we must remember that if either of the outer layers of the skin are opaque to light of any particular colour, then light of that colour cannot be scattered by the deeper layers, since light of that colour will never reach the deeper layer.

The problem we have to consider then, is the effect which various changes in the three optical layers of the skin will produce on the light scattered by the skin back to the eye of the observer, and more especially why there is less red light scattered by the skin of Europeans in the tropics than by the skin of a European living in Europe.

It will be convenient to consider the deepest layer of the skin first, *i.e.*, the layer with blood capillaries. The red colour of the skin depends mainly on the amount of red light which reaches this layer, and is scattered by it back to the eye of an observer. A decrease in this will be brought about either by a decrease in the quantity of blood in the capillaries or by a decrease in the quantity of red light, which gets through the epidermis to this layer. I argue that there is no decrease in the quantity of blood in the capillaries, hence the white colour of tropical skins must depend on the smaller quantity of red light which reaches the capillaries, and is scattered there back to an observer's eye.

A decrease in the quantity of red light, which reaches the capillary layer, can be caused in three ways :—

1. There may be a greater thickness of the unpigmented epidermis,

which (unless the epidermis is perfectly transparent) will allow less light of all colours to pass through to the capillary layer, and will, at the same time, scatter more white light back to an observer's eye.

2. Without change in the thickness of the unpigmented layer of the epidermis, it may become more milky, so that, as before, less light reaches the capillary layer, while more white light is scattered back to an observer's eye.

3. The epidermis may become partially opaque to red light by the deposition in it of a pigment opaque to red light.

We may sum up by saying that the anæmic appearance of the skin of a European in the tropics must be due to the epidermis becoming either thickened, more milky in colour, or more opaque to red light. I have no doubt but that variations in the thickness and milkiness of the epidermis do occur under different circumstances, and that these factors do influence skin colour; but the third factor, opaqueness to red light, is, I believe, the predominating factor.

Let us assume that the epidermis becomes partially opaque to red light. Then the light which is scattered by all except the most superficial layer of the epidermis will be deficient in red rays. But if this deficiency is just made up for by the red rays which are scattered by the blood in the capillaries, the skin will appear white in colour. This is, I believe, the true cause of so-called tropical anæmia.

In point of fact, I do not suppose that it is the red light only which is absorbed by the epidermis; the blue, violet, and green are also absorbed to a greater extent than the yellow; but it is the relative opacity of the epidermis to the red rays of light which annuls the red colour of the blood capillaries, and causes the apparent anæmia. Of course, the only way the epidermis can become partially opaque to light of any particular colour is by the deposition of pigment in the epidermis. In other words, an anæmic look coincides with the very first stage in the production of pigment in the epidermis. Such production of brownish pigment is, of course, admitted by everyone in the case of dark races, and also in the case of Europeans who are much exposed to the sun: in the case of Europeans the resulting colour is usually complicated by "erythema solare."

There is one factor which for simplicity I have left out of the above discussion. The appearance of the skin may vary according as to

whether the pigment tends to be concentrated in the more superficial or in the deeper layers of the epidermis, unless, indeed, the epidermis can be regarded as perfectly transparent. If the pigment is concentrated in the superficial layers, little light will penetrate the epidermis, and little light will be scattered there, and the skin will appear black. If the pigment is concentrated deep in the epidermis, and if the superficial layers are able to scatter light, the skin will then appear of a lighter colour.

There are certain well recognised instances when white light, or a white colour, is made from coloured light by absorbing the colour which is in excess. For satisfactorily viewing microscopic slides stained with hæmatoxylin and eosin, white light is required. An oil lamp gives out yellow light in excess, and, in consequence, we insert a blue glass, which is relatively opaque to the yellow rays of light, and, as a result, we get a whiter light. Again, linen, after use, tends to become tinged yellow, and a good laundryman knows that he can correct this by lightly staining the articles with "blue," which is relatively opaque to yellow light In the case of a European's skin it is the red rays which are in excess. In the tropics these are absorbed by a small amount of pigment in the skin, and so the skin appears whiter. When the pigment appears more plentiful the skin, more or less, assumes the colour of the yellow-brown pigment or pigments.

The change which takes place in the skin can also be imitated in the following way. If a deep-red coloured piece of paper, such as the 100 per cent. red on a Tallquist hæmoglobin scale, is placed on a table and looked at in a strong light through a piece of slightly transparent paper, the red tint will be visible, just the same as the red tint of a European's skin is visible. But if we insert between the red paper and the slightly transparent white paper a piece of black paper, or a piece of paper opaque to red rays, the red tint will be removed, and we shall only see the white or grayish colour of the slightly transparent paper.

A METHOD FOR THE TRAPPING OF *GLOSSINA MORSITANS* SUGGESTED FOR TRIAL

By J. O. SHIRCORE,

Medical Officer, East Africa Medical Service.

It is not proposed to discuss at length why the method described below may be useful; a short outline is sufficient to indicate that such a method may be worthy of trial. It is based on the known habits of this tsetse, described and reiterated time and again for many years.

(a) Attraction for colour: dark colours, such as dark blue, dark gray, etc.

(b) The attraction of the fly by moving objects. The light structure and the shape of the traps are particularly designed to catch any prevailing wind, in order that the traps may be thrown into movement.

(c) The "following range" (Dr. HODGES).

(d) The habit of *G. morsitans* of indulging in short periods of intermittent flight and rest.

(e) The dispersal of the fly when the object they are following comes to rest.

(f) The fact that female *G. morsitans* are more frequently taken on moving objects than otherwise (Sergeant-Major GIBBONS).

(g) The large area of adhesive surface exposed: the diagram shews forty traps, which represent 240 square yards.

(h) The marked preference for forest in contrast to their dislike for open plains and cleared spaces.

(i) The concentration of *G. morsitans*, for various reasons, at certain times into definite areas during the height of the dry season, August and September, in the Nyasaland Proclaimed Area.

(j) That no special decoys are necessary, insomuch that the natives of the district would act as such while travelling. The traps could be placed at suitable positions, so as to act as "interceptors." The space between the traps—*i.e.*, one yard in every direction—is sufficient to allow of free passage through these systems. Men or animals may be used as decoys to entice the fly into these situations, and a slow pace should

be observed while going through, if not an actual halt in the middle for a short while.

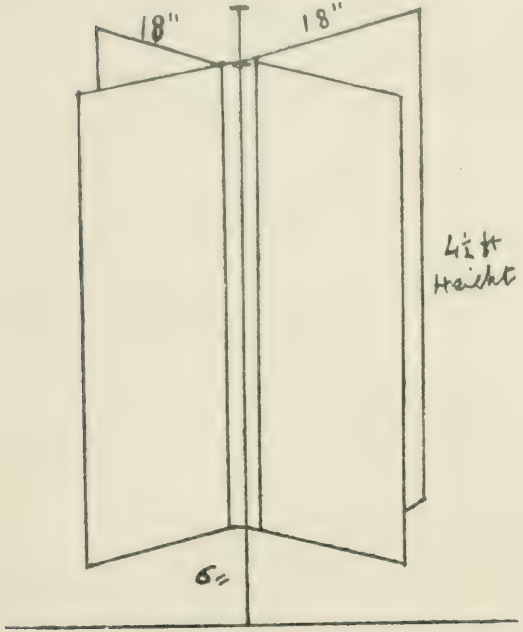
(k) That this method may possibly be found of value when employed for the purpose of controlling the extension of fly-belts along traffic routes into fly-free country.

The trap I suggest may be best made of light canvas, in lengths of 4½ft. by 1½ft. wide, and of any suitable dark colour. The ends of the canvas sewn—*vide* diagram No. 3—so as to slip over light rods of iron. Four pieces of canvas to go to one trap, and the surface adhesive on both sides: the capturing area of each trap being six square yards. The light rods should be placed at right angles to each other, top and bottom, and adjusted so as to pivot round on the central support, which must be stout enough to prevent the trap from being blown over. About six inches should be allowed between the lowest part of the canvas and the ground level.

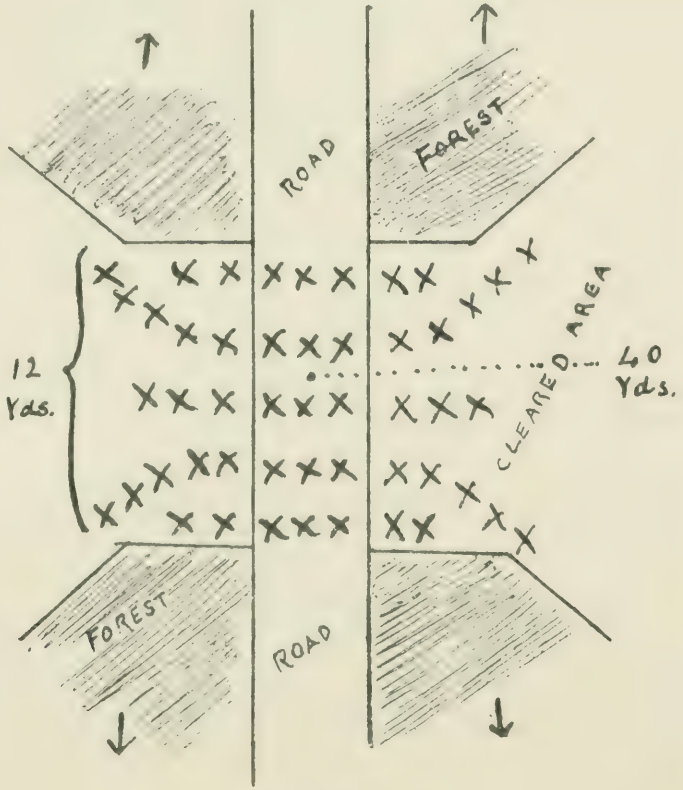
The arrangement of the traps can be modified as desired, and the most useful formation discovered by experiment, and added to or reduced in number; but the distribution demonstrated in diagram No. 2 may prove to be a good model to begin work upon.

Before the traps are erected an area should be thoroughly cleared, as indicated in diagram No. 2. I would suggest forty yards on each side, measured from the centre of the road or path, and not much more than twelve yards for the length of clearing for the trap-covered area; if too much is cleared all the fly may not follow through. The shaded area in diagram No. 2 is forest. Numerous suitable sites exist in the Proclaimed Area, Nyasaland, and one in particular situated just before the path from Kambwiris village leads on to Kuti Dambo.

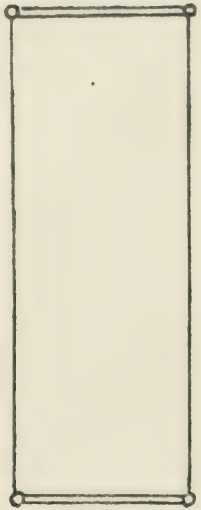
Very little clearing would be required in some places, and the whole experiment would not cost more than a few pounds.



DIA. No. 1.



DIA. No. 2.



DIA No. 3

Diagrams of Traps described by Dr. J. O. SHIRCORE.